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PHYSICAL EXAMINATION OF WEAK CHESTS

AND

DIFFERENTIAL DIAGNOSIS OF THE SEVERAL FORMS OF EARLY PHTHISIS.

BY

E. DARWIN HUDSON, JR., M.D.,

Professor of General Medicine and Diseases of the Chest, New York Polyclinic, Physician to Bellevue and St. Elizabeth's Hospitals.

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New York:
STYLES & CASH, Printers and Stationers, 77 Eighth Avenue.

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OUR system of physical examination of the chest, as developed by Avenbrugger, Laennec and Piorry, has received numerous amendations and additions from a host of investigators and teachers in Europe and America. The more prominent of these abroad are Skoda, Allison, Gairdner and Walshe; and not less valuable and original are the contributions in our own country, of Flint, Loomis, Leaming, Camman and Clark.

The profession has come to regard physical diagnosis, in the hands of experts, as affording exact knowledge of the conditions of the intra-thoracic organs. It is accepted, as a fact fully established, that, by the application of acoustic laws, certain combinations of physical signs lead to an immediate and correct diagnosis in most cases of pulmonary disease.

But the interpretation of physical signs can never be dissociated from some theory of the nature of the existing disease, a conception of its pathogenesis, and a knowledge of its gross and minute pathology, as well as of the pathological processes which determine its consecutive steps or stages, and its periods of transition from one to another form.

The conclusions of Piorry in percussion and of Laennec in auscultation were interpreted therefore, so far as regards pulmonary phthisis, upon the basis of the pathology of Bayle and Louis, the unity of pulmonary phthisis, and the tubercular nature, *ab initio*, of each and every case.

This view of the specific nature of all phthisis and its transmission solely by heredity had not been questioned for nearly fifty years.

But the last twenty years have witnessed almost a revolution of opinion as to the prominence, or causative relation, of miliary tubercle in phthisis. It may at least be affirmed, that, while division of opinion still exists on this question, a majority of all investigators and of the profession have come to acknowledge the three forms of phthisis—the catarrhal, fibroid, and tubercular—as so clearly presented by Sir Andrew Clark abroad and Prof. Loomis in our country.

But there is also reason to believe not only that pulmonary phthisis presents these three distinct types, the catarrhal or bronchial, the fibroid or interstitial, the tubercular or constitutional, but also that, in very many cases, it exists in a mixed form. A vertical section of many phthisical lungs, presents, in irregular distribution, areas of bronchial catarrh, thickened bronchus, peribronchial infiltration, bronchiectasis, interstitial fibroid infiltration, caseation, softening, antrum, and inflammatory changes in the investing pleura, not infrequently extending to the parietal pleura ; with intervening intra-pleural adhesions. It is not desirable to be diverted from the subject of our consideration, the physical diagnosis, to consider the many unsettled points as to the relative influence of bronchial, interstitial and pleuretic inflammations in developing this mixed pathological result.

But I am satisfied that, in consideration of the different methods of origin and the complex nature of a large proportion of cases of phthisis, our physical exploration is incomplete if we discover only the presence of cavities and softening, or the evidences of local consolidation. A complete diagnosis should include, *in extenso*, an exact knowledge of the condition of each portion of the respiratory apparatus, including the air passages, vesicles, interstitial tissue and pleura.

Under these circumstances, it is evident that the chapter on Physical Examination of the Chest, if by any regarded as closed, must be reopened to admit new facts and new interpretations of the old acoustic laws, in the light of a broader and changing pathological view.

The views which I present relate to the methods of examina-

tion most useful in studying weak chests with tendencies to phthisis, in detecting the times of transition from danger to disease, and in differentiating the early forms of phthisis. They include no new theory as to the disease, nor are they the product of speculation, but represent the opinions and convictions which I have reached, first as a faithful student and observer of our several authorities on the subject, and concurrently, by independent observation in private practice during the past eighteen years, and my clinics during the past twelve years, either at the Woman's Medical College, or at the New York Polyclinic and my wards in Bellevue Hospital.

I recognize with Simon, of London, that struma or the lymphatic temperament supplies a ready soil for phthisis ; with Niemeyer, Rindfleisch and the Germans of the past twenty years, that the lymphatic capillary tissue of the lungs is essentially the channel of infection for tubercular cases, whether by vitiated air and food, by toxic substances, or absorption of the liquified cheesy products of inflammation. But observation has also made me a firm believer in the agency of inflammation as the chief factor in developing pulmonary phthisis, whether in those predisposed by heredity, or in those free from such predisposition ; the latter class, I believe, to include the great majority of cases. That inflammation, in intercurrent attacks, is the most prominent feature of the progress of phthisis from stage to stage is quite generally recognized.

In our conception of phthisis, it will be of advantage to defer the study of the disease in its mixed and complex forms, until the essential evidences of the three definite types are reviewed. Virchow has exerted a powerful influence in favor of keeping close to anatomical and physiological purposes of parts in the study of their morbid changes, calling attention to the fact that in most organs disease is primarily either of its functional structure, or of its interstitial vascularized parenchyma, or of its cortex and investing capsule. It is so with the meninges of the brain and cord, the nerve substance and connective tissue. So also in the liver, and in the kidneys. Glomerular nephritis, tubular nephritis and

interstitial nephritis, start independently, but ultimately become more or less associated in the same organ.

In the thorax, with reference to the respiratory organs, this is especially true. The larger bronchi have a set of afferent and efferent nutritive vessels essentially separate from those of the infundibuli and air sacs, and the latter, while receiving in a limited degree the blood of intervening bronchial arterioles, yet possess a circulation of their own, and the circulation of the parietal pleura is wholly independent of either. We are prepared, therefore, in advance of actual search, to find diseases distinctly originated in these differently vascularized tissues and regions.

An epitomized statement of the essential physical signs of early phthisis, as generally held, may be included under two heads:

1st. That the existence of an area of dullness on percussion, of absent or enfeebled vesicular murmur, with increased conduction and raised pitch of respiratory, voice and tussal sounds, indicates parenchymatous infiltration, whether of tubercular or inflammatory origin—the air sacs are either collapsed or engorged with catarrhal products—in fact a consolidation existing, which tends ultimately to caseation, softening and cavity. Local subcrepitus in such an area is regarded as evidence of collateral hyperæmia, or, of the existence of a local catarrhal process, tending to further interstitial deposit and further occlusion of air vesicles. The history of the patient influences the diagnosis as to the tubercular or catarrhal nature of the case.

2d. That an area of notable dullness or flatness on percussion, and of deficient conduction of sounds points to fibroid phthisis. This deficiency of sounds may range in degrees from enfeebled sounds, though masked, distant, indefinite sounds, to silence, or, with the deficiency of conduction of the breath, voice and cough characterizing fibroid, may also be associated other diagnostic adventitious sounds, as of emphysema, bronchial constriction or bronchial cavity.

In addition to the above recognized distinction, I believe that many careful students of chest acoustics have come to a recognition of the differentiation of the forms of fibroid consolidation,

whether starting from the bronchi, or centric and diffuse, or again distinctly of pleural origin. Thus,

(*a.*) When over a definite area the percussion note is pronouncedly flat or high pitched, with relatively but a slight conduction of the normal sounds, unchanged in pitch and quality, and when the sounds, though discernable, are feeble and distant, the fibroid is likely to be peripheral, starting from the pleura, the product of an imperfectly resolved pleuro-pneumonia, or of a local plastic pleurisy leading to underlying hyperæmia and subsequent hyperplastic fibroid.

(*b.*) When the union of flatness on percussion and obscuration of respiratory and voice sounds is pronounced, especially if bilateral and attended with great reduction of respiratory motion on both sides, the fibroid is likely to be more generally and diffusely interstitial. Such is the condition found in advanced cases from the gouty diathesis, in brown induration or interstitial results of long standing cardiac incompetency, and in syphilitic phthisis. The same conditions existing in but one lung cannot be inferred with equal certainty.

(*c.*) When the percussion note, relatively, is but slightly raised in pitch, being rather deficient in duration, and devoid of pulmonary quality, and there co-exists a general enfeeblement of respiration, or a general absence of volume in respiration, the fibroid is likely to have started from the bronchi. The presence of an exaggerated tubular quality of inspiratory sound, with slight increase of pitch ; of retarded tubular expiration, with or without rules, will further determine whether the case is more specially one of peribronchitis, or bronchial thickening, or bronchial rigidity and dilatation.

The first point which I desire to discuss, with reference to efficient and correct chest exploration, is the importance of examining all parts of the chest. Too often the front of the chest is cursorily examined, with reference chiefly to the condition of the apices. The chest should be divested of clothing, or, in the case of ladies, covered only with a thin merino shirt or dressing sacque, which may be raised in parts as required for inspection of the

chest wall. The chest should be examined from apex to base of lungs—front, side, and back. Two areas, almost wholly passed over by most examiners, often afford most valuable information. They are : first, the high axillary region, and second the interscapular spaces.

The examination in the axillary line high up, with the arm carried over the head, should be made in all cases. It must have come to the notice of very many physicians in private practice, but more often in the grouped cases of general hospitals, that in cases of irregular pleuro-pneumonia, and in low and indefinite forms of pneumonia—the product of squalor, dissipation, malaria or secondary to phthisis—the initial area of pneumonic consolidation, as evidenced by bronchial breathing, has been found only in the axilla.

The importance of the interscapular exploration is such as to warrant a fuller consideration. It is here that we have the larger bronchial trunks and the hilus of each lung close to the posterior wall of the thorax. But in the ordinary postures, with the head erect and arms hanging by the side, or even with the arms loosely folded in front, the interscapular spaces are relatively valueless for physical diagnosis. The scapula, on either side, covers the chest from the first to the eighth rib, and comes within an inch of the processes of the vertebræ. This intervening space on each side of the spine is filled in deeply by the rhomboideus minor and major, which span it obliquely from the vertebræ to the scapula, and to complete the muscular envelopment the trapezius extends from its median origins at the occiput, the ligamentum nuchae the cervical and twelve dorsal vertebræ, in converging bands, over the interscapular space and scapular blade, to its attachment on the spine of the scapula. The value of the upper half of the back for percussion is therefore slight, over these thick flaccid muscles and this interposed bony plate. Most physicians recognize this condition and obviate it, in a measure, by directing the patient to fold the arms. By a forward motion of the arms, the claviculo-scapular union, being essentially fixed, serves as a point of rotation, while the tension of the deltoid on the spine of the scapula directly

antagonizes the trapezius and draws the scapula laterally with slight rotation. Dr. Corson, more than any other person, has emphasized the importance of taking advantage of the great mobility of the scapulæ as a means of increasing the inter-scapular area.

If the arms be crossed in front, each upward, at an angle of 45 degrees, and the hand on either side is made to grasp the convexity of the shoulder, the clavicular attachments and the deltoid so elevate, rotate, and draw outward the scapulæ, that the inter-scapular space is increased more than two fold, and the previously flaccid and thick muscles are rendered tense and thin, lying firmly on the ribs, and interposing no obstacle to percussion and auscultation of the bronchi, and of the upper inner borders of the lungs. Each intra-scapular area, before a rectangle of one inch width, extending from the first to the eighth rib, is converted into a trapezoid whose upper truncated apex is fully two inches wide and whose base is between three and four inches broad. The lower angle of the scapula is elevated to the seventh rib. This great gain in space, however, is not more important than is the advantage from the thinning out of the trapezius and rhomboidei. Finally, if the patient's head be bowed, and the neck forcibly incurvated forward, the tension of the levator anguli scapulæ is added ; it increases the elevation of the scapulæ by half an inch, and adds to the general tension of the dorsal muscles. The scapulæ have not only been moved laterally, but the thin plates, as located, are firmly held between tense muscles, and interpose a greatly diminished obstruction to conduction of sounds. I would urge the resort to this position as greatly facilitating the study of chronic bronchitis, peri-bronchial fibroid, and, indeed, of all forms of bronchial and pulmonary diseases.

In treatises on the systematic exploration of the chest, "inspection" is, by all authors, placed first ; but a variable and limited value is ascribed to it. I am, however, convinced that in a large proportion of chest cases, and in a large number of phthisical ones, an approximate diagnosis can be made by inspection alone.

The second consideration of this paper, therefore, is the

diagnostic importance of inspection, and the desirability of extending it, in detail, to all the structures of the thoracic wall. It is customary with all to observe certain general features by inspection, as the breadth of the shoulders, the equal or unequal volume, and the symmetry or asymmetry of the two sides, the relative height of the shoulders, the comparative fullness of respiratory expansion, areas of bulging or retraction, and the relative prominence of the sternum and costo-cartilaginous unions. But three other points, in inspection, of much value, may be briefly discussed.

The first is the direction of the clavicle. Broad shoulders and a nearly horizontal inner half of the clavicle give promise of fully developed lungs. The clavicle in some persons is horizontal in its inner half, though more often it is deflected upward from 5 to 10 degrees, when it curves, going more obliquely upward and backward in its second half, to its scapular attachment. The upward angle of the line of the clavicle as a whole, is, therefore, in healthy chests, within the range of 10 to 15 or 20 degrees. But when the clavicle is deflected upward and backward, at an angle of 30, 35 or 45 degrees, the chest development has manifestly been most defective during a long term of years, and the presumption is in favor of the existence of lungs, undeveloped at their apices, which have never fully occupied the supraclavicular and clavicular areas.

The second point is with reference to the relation which slight lateral curvatures of the spine bear to the condition of the respiratory organs.

An inquiry into the history of a considerable number of cases of lateral curvature will elicit the fact, that its appearance has been subsequent to the occurrence of a pronounced attack of pleuritis or pleuro-pneumonia, or a period of long-continued unilateral thoracic pain with associated cough. It is further found in many of these cases, that the deflection is toward the side of the chest possessing most respiratory expansion, and that the concavity of the primary curve, and the convexities of the compensatory curves are toward the side presenting slight or

notable diminution of respiratory motion. And further exploration, by means of percussion and auscultation, establishes the fact that the curved section of the spine lies within a zone of the chest, affording evidences of thickened pleura, fibroid phthisis or peribronchial consolidation. Where this combination of conditions exists, it is reasonable to conclude that the intrathoracic changes, of one side, have diminished the respiratory action of the overlying muscles, and that an unequal traction of the spine, by the antagonizing muscles of the two sides on the back, has resulted in the curvature to the side of greater respiratory action and of stronger muscles. I do not mean to imply that any proportion of lateral spinal curvatures are a sequence of pulmonary disease, but to emphasize the fact that this is the case sufficiently often to warrant inspection of the spine in all cases.

Barwell has called attention to this fact in so far as it relates to the etiology of the spinal disease. Reversely, it may be noted that pronounced lateral and even angular curvature, originated through dyscrasia or malnutrition, and of long duration, does not materially cripple the contained lungs. From the examination of a considerable number of chests, seriously distorted by spinal disease, I have been surprised to find how generally the lungs and heart have adapted themselves to their changed accommodations. They are often exceptionally healthy, and when otherwise, instead of inflammatory changes, or phthisical deposits, we find emphysema alone has been developed as the result of the chest asymmetry.

The third and final consideration which I present, under inspection, as pertaining to early phthisis, is the importance of closely observing and interpreting, with their full significance, the changes in the individual intercostal spaces and also in the supraclavicular space, suprasternal notch, and epigastrium.

A pronounced depression above and below the clavicle, associated with narrow shoulders and marked deviation of the clavicles upward and backward, may exist over feebly developed apices, which, however, are not diseased. But such depression, in a person possessing ordinary breadth of the shoulders, and whose

clavicles are but slightly deflected upward, is indicative of consolidation of the apex, whether by tuberculatization or inflammatory infiltration. In both cases little respiratory movement will be observed, but percussion and auscultation at once determines which of these two states exists. The sinking in of the infraclavicular space, or of special intercostal spaces, synchronous with the inspirations, and notably in chests which, as a whole, retain their full expansion, points definitely to the existence of a peripheral fibroid of the lung, originated by plastic pleuritis or pleuropneumonia, and suggests adhesion of the opposed pleuræ beneath the points of depression.

When, however, the chest expansion is visibly diminished or suspended—the bony thorax being in a state of fixation—inspiration becomes essentially diaphragmatic Under these circumstances, sinking of the suprasternal notch, the supra and infraclavicular and intercostal spaces, and of the epigastrian, synchronous with inspiration, points to a more diffuse and deeper fibroid, shooting from periphery to center of the lungs, and drawing not only upon the interspaces of the chest wall, but also drawing upon the walls of the bronchi, and developing bronchiectasis. Again, resort to percussion, and auscultation will verify these conclusions.

These fibroid areas and bronchial distortions cannot be dissociated from the study of phthisis; they form a part of it, since they determine the recurring hyperæmia and catarrhal processes, which terminate in inflammatory infiltration and ultimate caseation.

One further phenomenon of the intercostal spaces, of exceptional occurrence deserves mention. It occasionally happens in an emaciated chest, that the cardiac impulse, instead of presenting only the usual apex beat, perceptible in the fifth intercostal space, is equally distinct in the fourth, third and second incostal spaces. These interspaces to the left of the sternum are seen to rise and fall, synchronous with the action of the heart. The inference is plain, atrophy and retraction of the thin borders of the left lung, which usually overlap the heart, have taken place.

This condition, as a rule, signifies the existence of advanced phthisis of the lung above and adjacent to the heart; and often there is a large antrum and collapsed lung; but I have once found it, where fibroid contraction only existed in the adjacent lung. The diagnosis of this condition is confirmed by percussion, since the normally small triangle of superficial cardiac flatness is increased to a large circular or irregular area, and further verified by auscultation, since all respiratory and conduction sounds are wholly removed from this area. I am not prepared to say, from the limited number of cases I have seen, whether this cardiac motion, visible in all four intercostal spaces of the præcorcial area, warrants an inference of pleuro-pericardial adhesions. The cases I have seen have given no history of previous pericarditis, nor of local pleurisy.

The retraction of an atrophied, cirrhosed lung establishes direct contiguity of the anterior cardiac surface and the chest wall. The interposition of pericardial or even of intrapleural bands, therefore, is not essential to the production of this disseminated heart impulse, and we know that none such is required to transmit the localized normal apex beat.

Before dismissing the subject of chest inspection, I desire to state my views as to the causative relation of the non-development of the chest wall and its movements to phthisis. It is well known that the apices of the lungs are the most frequent site of early phthisis. Yet nothing peculiar in the anatomical structure of the apices, or in their function, explains their greater liability to disease. Women are more prone to phthisis than are men, yet women have a greater development of upper costal respiration, or apex expansion, than do men. I believe that phthisis attacks the apices preferably, because,

1st. The apices are so frequently enfeebled by contraction of the upper part of the bony thorax, whether by want of muscular activity and development or by rounded shoulders, through carelessness and indolence, or through confining work at desk or trade.

2d. That the apex region in this enfeebled condition is soonest

reached and most easily invaded by the frequent recurring catarrhal inflammations descending from the larynx and trachea.

3d. Because the apices, when invaded by hyperæmia or catarrhal processes, are not favorably affected by the energetic force of cough.

Cough is essentially a diaphragmatic act; it expends its force upon the lower two-thirds of the lungs, tending to clear the lower bronchi of contained mucus, and by compression of the parenchyma to remove their hyperæmic conditions. But this benefit is not extended to the bronchi and parenchyma of the apices. It is a fact that in cough, the glottis being in a measure constricted, the forcible currents of air, driven upward from the lower portions of the lungs by diaphragmatic force, are not permitted to wholly escape through the larynx, but are deflected into the bronchi and vesicles of the upper lobes. When this process continues too long, as in pertussis or chronic cough, from any cause, emphysema of the apices or upper lobes (obstructive emphysema) results. In the same manner I believe that cough by pneumatic pressure frequently detains or impacts catarrhal products in the tubes and vesicles of the apices, and renders them especially the seat of phthisis.

I shall not dwell on mensuration, since its essential features are established, and it affords but little diagnostic information. Asymmetry of the two sides of the chest, or a marked diminution of respiratory expansion are to be interpreted subsequently by percussion and auscultation.

Palpation as a method of chest examination, while presenting many special details, is particularly valuable as relates to the subject of fremitus. Fremitus is especially instructive for negative diagnosis, often indicating the normal chest by a characteristic vibration communicated to the hands, which they cannot misinterpret. But fremitus may be feeble or almost absent in healthy but indifferently developed chests, especially if the patient's voice be treble. Fremitus may be weakened by rigidity of the bronchi. Fibroid areas may remove it, in whole or in part. Thickened pleura may remove it, or rarely increase it by conduc-

tion, depending upon the condition of the bronchi and pulmonary tissues. Finally the possibility of fremitus under these and all conditions depends largely upon the elasticity of the ribs. When by any cause, whether by senile calcification or by the inspiratory fixation of the thorax in emphysema, the ribs are held tense and rigid, fremitus ceases, no matter what the condition of the contained organs. Palpation therefore of itself does not afford valuable diagnostic information as to the state of phthisical lungs.

Although most authors and teachers of physical diagnosis recognize the composite nature of the pulmonary percussion sound, and the several factors which unite to produce it, yet the impression still prevails that it is essentially the product and exponent of the underlying lung. It would be undesirable to detract in the slightest degree from the confidence reposed in the diagnostic value of percussion. But the exclusively pulmonary origin of percussion sound is easily disproved. This is most easily accomplished by a brief reference to the percussion note of emphysema. If, in emphysema, the percussion note were to correspond to the volume of air within the coalesced air-vesicles, and in the stretched infundibuli and dilated terminal bronchi, we should obtain by percussion the prolonged, low-pitched, tympanitic sound which the novice in physical diagnosis is always prepared to hear. To his surprise, the sound, although possessed of some tympanitic quality, is curtailed in duration, and in its initial portion, at least, is raised in pitch. The explanation is simple. The emphysematous lung is an aggregation of fused air-vesicles and distended terminal air-passages; it is a dilated lung, with much of its elastic tissue absorbed and deprived of its expiratory contractibility. The overlying bony thorax is, therefore, in a state of inspiratory fixation, and so tense or rigid that it does not vibrate. Hence the contact of the percussing finger tips with a rigid thorax elicits a sound relatively high in pitch and devoid of vibration or duration. I will mention one further fact bearing upon this question. The middle and lower sternal regions overlie the anterior mediastinum, which contains the great vessels and also cover the body of the

heart. Yet, in some elastic chests, percussion over the lower half of the sternum—the keystone of the elastic respiratory arch—elicits, not flatness, the product of underlying solid structures, but a sound essentially of pulmonary quality. The elasticity of the chest wall is undoubtedly an essential factor of, or contributor to, the percussion note.

However, it may be affirmed that raised pitch of the percussion note, especially at the apices, most frequently indicates lung condensation, either tuberculartzation or inflammatory infiltration. It may also mean thickening of the underlying pleura. But, on the other hand, when the chest wall is poorly expanded, whether by disuse and enfeeblement of the upper respiratory muscles, or when fibroid phthisis or bronchial rigidity have transferred respiratory action to the diaphragm, then the motion of the ribs lessens, and the disused ribs become more or less fixed. Under these conditions, percussion, over the entire chest, frequently elicits only an unsatisfactory and negative sound, devoid of diagnostic features.

Reversely, in many cases thickening of the pleura, of recent or remote development, may cause such absolute percussion flatness as to warrant the inference of a solidified, phthisical lung. We may conclude, therefore, that in contracted chests and in the mixed forms of early phthisis, percussion leads to no positive diagnosis, until supplemented by auscultation.

But there is yet another physical sign, or differential test, to which I must call special attention. It may be regarded as a product of mediate percussion. It is in no wise connected with chest acoustics, but rather pertains to the subject of the thoracic innervation and the influence of pulmonary tissue waste upon the spinal centre. I refer to a special cutaneous reflex, which characterizes many, perhaps most, phthisical cases. It is so frequently present at the periods when caseation and secondary inflammations mark a case, hitherto uncertain, as one of phthisis, that it be regarded when present as pathognomonic. As early as 1830, in the Dublin Hospital Reports, Vol. V., p. 70, Graves and Stokes described certain muscle tumors which appeared following direct

percussion upon the chest "in incipient phthisis chiefly over the seat of irritation." Dr. Warburton Begbie observed and taught it as an evidence of phthisis. Dr. Lawson Tait, in 1871, in the Dublin *Journal of Medical Science*, Vol. LII., p. 316, described this phenomenon and designated it more classically as "myoidema," or muscle tumor. Prof. Gairdner and Dr. Finlayson, in the work of the latter, also allude to the diagnostic value of the sign, and properly add that "myoidema" and muscle tumor are misnomers, since this reflex is *of the skin alone;* that it is analagous to the contraction of the dartos of the scrotum; that it is best produced by quick strokes of two or more finger tips upon the costal cartilages, and is much less marked over the muscles of the intercostal spaces. I should hesitate to call, anew, attention to a point advanced so long since by Graves and Stokes, and which presumably had long since passed to oblivion, because tried and found devoid of significance, were it not that I have first carefully studied it upon several hundred chests at Bellevue Hospital and the Polyclinic. Being myself skeptical of its special relations to phthisis only, I have at the outset asked myself, as most of my students in physical diagnosis have asked me, if it was not present in all cases of emaciation, wasting disease, and slow convalescence. Hence I have sought to find it in the chests of convalescents from protracted and depressing typhoid, in advanced cirrhosis, in abscess of the liver, in hemiplegics and paraplegics, and, indeed, wherever disease had produced tissue waste. The result of my observations was, first, that it is usually present in established phthisis, where the long-standing and progressive intra-thoracic disease has wasted the body; second, that it frequently exists in incipient phthisis; third, that it is exceptionally present in any other disease, and its presence in any such case justifies a suspicion of phthisis. In but one case, a convalescent typhoid, did it seem pronounced, and so exceptional was this case that though chest disease was not demonstrable, I felt that its early manifestations might soon appear. Again in cases of chronic bronchitis, bronchial dilation, and bronchiectasis, it does not appear, nor again in cases of thickened pleura, however extended;

but when fibroid is developed, whether of bronchial, interstitial or pleural origin, so as to contract the chest and to cripple the lungs for respiratory service and lessen the nutrition of the body, it begins to appear. It is most pronounced when elevation of temperature, colliquitive sweating and bodily debility indicate pulmonary tissue waste and caseation In advanced phthisis, with softening, cavity and excessive muco-purulent sputa, it reaches its maximum. I usually percuss with a quick firm blow, with two or three finger tips of the right hand, upon the costal cartilages of either side. Instantly two or three little conoidal tumefactions appear, rising to a variable height in different cases, ranging from one-sixteenth to one-eighth, or rarely one-fourth of an inch. Remaining fixed for a variable period, rarely so brief as a second, more often two or three seconds, they undergo a subsidence more gradual than the elevation, a vibratile, vacillating, interrupted recession. Graves and Stokes recorded a duration of four seconds and Tait one of twelve seconds. Finlayson speaks of it as "a peculiar momentary starting and elevation of the skin." Over the intercostal muscles it also can be produced. I have sometimes seen in the intercostal space an associated reflex muscle wave which floated away towards the shoulder until lost ; but this muscle reflex is not the peculiar reflex which has come to be known as myoidema, which is peculiarly a cutaneous reflex, a contraction of the arrectores cutis. It is not to be confounded with reflex of the thoracic muscles, any more than should cutis anserina, suddenly following a chill, or urticaria following irritation of solar heat or indigestion. In some well pronounced cases of phthisis I have been able to develop myoidema behind in the suprascapular space, but especially in the interscapular space when broadened by Carson's shoulder position. Myoidema then is a cutaneous reflex. "Reflex hyperkinesis," we are told by Ross, "is caused by disease which increases the irritability, and consequently decreases the specific resistance of the reflex arc in any part of its course," etc. I have, therefore, so far as I have studied myoidema, come to regard it as of diagnostic value. I regard it a corroborative sign of existing phthisis, and as indicating the arrival

of a case of phthisis at a point where the nervous system has become weakened, the vitality depressed beyond the point of resistance, and as marking the case as one tending toward an unfavorable result. If found in a case upon which I had hitherto looked hopefully, it would lead me to renew my examination for evidence of infiltration and caseation. I would ask observers of the chest to habitually apply this test and add their contributions as to its value or uselessness. At present, I am disposed to regard it as worthy to be added to the associated signs of pulmonary phthisis.

We come, finally, to the auscultatory evidences of pulmonary phthisis. Enfeeblement of the vesicular murmur, and its entire absence, have long been regarded as evidences of condensation of lung tissue, even when conduction sounds have not been raised in pitch; and when to absence of vesicular murmur are added bronchial breathing and broncophony, no doubt is left of the existence of an underlying area of infiltration. Were all phthisis, either *ab initio*, or even ultimately, tuberculariza tion, or even disseminated interstitial infiltration, of inflammatory origin, we might need no further combination of physical signs than absence of vesicular murmur and conductions of sounds raised in pitch. But every practitioner, and every teacher of auscultation, has frequently been confronted with cases where he found nothing definite, or a most obscure combination of auscultatory signs, rendering the above tests most unsatisfactory. This is explained by the fact that in mixed phthisis a combination of peribronchial exudation of fibroid, emanating from the bronchi or the periphery, and of pleuritic thickening, exists to make up the structures upon the expansion of which the development of the sounds depends, and which are also the media for their conduction. With reference to vesicular murmur, even in health, I am not at all sure that the majority of ears can dissociate it during active respiration, on the one hand, from the finer tubular friction sounds, or, on the other hand, from the sussurus of the thoracic muscles. I am almost certain that the vesicular murmur, or true respiratory murmur, as claimed to be heard during suspended breathing, or the holding of the

breath at the height of forced inspiration—the suspension having in view to remove the tubular or friction elements of inspiratory sound—is often confounded with, or masked by, the muscular sussurus, which is, like respiratory murmur, so characteristically low pitched, multitudinous and diffused.

The absence or modification of the vesicular murmur has always and properly been regarded as a first warning of phthisis. But the presence or absence, fullness of development or enfeeblement of the vesicular murmur, is not of so great value in differentiating the three forms of phthisis, and especially the relative proportion of each in mixed phthisis. The crippling lesions, in many cases, have so contracted the chest, have so diminished its expansion, and lowered the velocity and force of the incoming inspiratory currents, that but little respiratory sound is developed to be conducted. Under this embarrassment, however—the absence of respiratory sound—the phonated voice supplies us with a sound produced quite independent of chest rigidity, diminished expansion, and enfeebled air currents. And thus, when considerable areas of pulmonary change exist, the voice, as it reached the ear, by its pitch and quality and degree of dissemination, tells of the media which have transmitted it. Thus the voice is high-pitched when conducted through tubercular areas, in proportion to the size of the area and density of the consolidation ; or it is rendered masked and distant in quality by transmission through moderate thickening of the pleura ; it is faint or absent in exceptional cases of very thick pleura, though *per contra*, in rare instances, a pleura of one-half or three-quarter inch thickness may conduct the voice sound with raised pitch. But the vibration of the bronchi, the lung parenchyma and chest wall, by the full voice, often gives no indication of the presence of the lesser lesions of early phthisis. The pronounced words, especially of low-pitched bass or baritone voice, are reverberated and resonated with such force in the bronchi that they are unchanged in quality, and are brought to the ear scarcely modified in pitch. Such is the case in slight interstitial infiltration, or a peribronchial thickening, or limited fibroid ; nor will slight thickening of pleura al-

ways modify the conducted voice, though the existence of such pleuritic lesion is evidenced by dullness on percussion. In such chests, then, in order to determine these lesser deposits, we still need, for conduction, a sound produced independent of the enfeebled chest expansion and slowed air currents, but more delicate and less voluminous than the voice. This we have in the whisper. I have for a long time come to regard the study and application of the conducted whisper as of equal or greater value than the study of changed vesicular murmur and conducted voice, and of far wider application in determining and differentiating the early steps of the three definite forms, and the many mixed cases of phthisis. 1st. The patient, directed by the physician, may utter a quiet, almost inaudible, low-pitched whisper, which in quality and volume supplies the place of conducted tubular and vesicular sound, and which latter are often absent in the contracted chests which we are considering; or, 2d, he may whisper clearly and audibly; or, 3d, he may utter a short, abrupt, forcible, high-pitched, aspirated whisper, deficient in volume and duration, but of great intensity, a sound which preserves its characteristics unmodified by slight changes of tissue density, and capable of transmission through bronchial thickening and fibroid areas, when the feebler forms of whisper, like vesicular murmur, would be unconducted and unheard. Thus, at the apex, the low-pitched whisper, changed by transmission through the pulmonary media to a high-pitched sound, is diagnostic of incipient infiltration. The non-conduction of such a low-pitched whisper—silence only—or the distant transmission of a high-pitched whisper, when associated with transmission of the voice, unchanged in quality and pitch, point to peribronchial thickening or centric fibroid of limited extent. But the non-conduction of both the low and high-pitched whispers, and a distant feeble conduction of the voice point to a fibroid nearer the surface or progressing inwards from a thickened pleura.

Finally, the ear, applied over the chest, often cannot detect differences in adjacent areas, as lobular consolidation. By the substitution of the smaller tube of the stethoscope, and uttering

the incisive, high-pitched whisper, close comparison of adjacent limited areas, as contiguous lobules, can be made to great advantage. Chest vibration—an objectionable element, diffusing and disseminating sounds—is excluded by substituting whisper for the too energetic voice, and very direct conduction, by each underlying area, is secured.

I also especially value the whisper tests, for so I have come to regard and call them, in distinguishing a healthy lung from one but slightly diseased—a question so often forced upon us by the existence only of failing health and narrowed chest. The low-pitched whisper possesses too little force to vibrate the healthy lung, or, reversely stated, the healthy lung is such a poor conductor of sound that the ordinary low whisper sound is dispersed in the ærated lung, and does not reach the chest wall and ear, or but feebly. As a negative sign, therefore, in examination of weak chests, it is of greatest value, and will rank as does absence of expiratory sound, as an indication of healthy lung.

The diagnostic significance of conduction of the tubular elements of respiratory sound, high pitched in inspiration, and prolonged and high pitched in expiration, needs no elucidation. When marked it is diagnostic ; but when it is not marked, when, as already stated, by reason of defective chest expansion, no bronchial tubular sound is developed, or it is developed but feebly, in that class of cases where the physician listens and hears little or nothing, the whisper still remains adaptable for each case and each form of phthisis, and is diagnostic. I have not alluded to the co-existence of cavernous breathing, retarded blowing, expiratory sounds, the presence of dry or moist râles, and the many other associated signs by which diagnosis is facilitated and confirmed, as I have not aimed to discuss the entire question of chest accoustics as applied to phthisis, but rather the methods which I regard most essential, including methods that are too little discussed and practiced.

A detailed examination in the light of our present knowledge of the pathology of phthisis, and by the systematic steps of inspection, palpation, percussion, and auscultation, even when supplemented

by the family and personal history, and the existing symptoms will, combined, in many cases, fail to lead up to a final diagnosis. Renewed and patient examinations will be necessary from time to time, under new circumstances of changing symptoms and pathology. The occurrence of an hæmoptysis is often the occasion of a first examination of a hitherto unsuspected or a long-neglected chest. The physical signs are widely different during the first hours or the days succeeding an hæmoptysis. At the time of its occurrence, gurgles and râles may mark the site of the gravitated blood, or possibly the area from which it escaped. Within a few hours arrest of hemorrhage and coagulation of the blood may remove these râles. Possibly a local area of muffling of all sounds, with or without dull percussion note, may mark the area of hyperæmia. In twenty-four or forty-eight hours the development of râles may give evidence of a local catarrhal process, or we may find the crepitus of a local pneumonic process. Finally, in some cases, no physical signs exist, the hemorrhage having acted to relieve a hyperæmic area.

One further and final point: In the study of incipient phthisis and weak lungs, no more important problem presents itself in exploration of the chest than the interpretation of local areas of subcrepitus, especially if co-existent with dullness on percussion and bronchial breathing, bronchial whisper, and broncophony. It was one of the errors of the past to interpret such subcrepitus in consumptive lungs as indicative of the process of softening.

We can pretty safely say to-day that there are no auscultatory evidences of softening, except those grosser and manifest sounds, the changeable râles and gurgles associated with forming cavity. The area of caseation and softening cannot originate sounds until communication with a bronchas has been established.

Subcrepitus, it is true, is often associated with softening, but is not in any sense the result of the action of respiratory currents on softening cheesy matter. Subcrepitus when local over phthisical areas, if low pitched and remote from the ear, denotes local capillary bronchitis, or more often hyperæmia and bronchorhœa, a transudation of serum or sero-mucus into ultimate bronchial

tubes. It is then a result of obstructed circulation and collateral hyperæmia. If, however, the subcrepitus be high pitched, brilliant, explosive, and manifestly directly under the ear, it is plainly inter-pleural. It then denotes that the infiltration of the lung has reached the surface, and given rise to secondary local pleurisy, or that the patient from exposure has contracted a local plastic pleuritis.

In all disease the effort of the physician should be specially directed to arriving at an early and accurate diagnosis. Prognosis and treatment cannot be of value except they be preceded by a correct conception of the pathogenesis of the malady and the departure from normal conditions which the affected structures have undergone. This proposition is regarded as especially true of systemic diseases the product of specific infection and sepsis. Yet these diseases often have a period of self-limitation, and progress by stages towards recovery, or are thrown off by an eliminative effort of the system, or can be successfully combated by specific agents, or rendered harmless, until their force is spent, by antipyretics and antiseptics.

But with reference to pulmonary phthisis, it may truly be said that the usefulness of the physician is greatest in the care and restoration of the enfeebled chest, and in the management of chests already invaded by one or other of the wasting processes.

The fully-developed consumption is so difficult to combat that it is an opprobium medicinæ that so little can be done. While then we are divided as to the pathogenesis, whether phthisis is disseminated through the lungs by the lymphatic capillaries; whether it is of inflammatory origin with subsequent sepsis by softened cheesy matter, or is specifically produced by colonies of bacteria, we may all agree that the suspected and involved areas should early be studied and differentiated.